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Early Development of Larval *Taenia polyacantha* in Experimental Intermediate Hosts

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ABSTRACT: The early larval development and migration route of *Taenia polyacantha* were examined using oral inoculation of oncospheres into red-backed voles and Mongolian gerbils. The larvae were recovered in the wall of the small intestine and in the mesenteric lymph nodes by 5 days postinfection (PI) and from the peritoneal cavity after 6 days PI. These results suggest that the larval cestodes developed initially in the wall of the small intestine and the mesenteric lymph nodes, and later migrated to the peritoneal cavity. Although the development of the parasite was quite similar in the 2 host species, pathological changes were different. In Mongolian gerbils, these changes were slight, but in red-backed voles, they were marked and fatal. In addition to oral inoculation, hatched oncospheres were injected intraperitoneally and subcutaneously into red-backed voles, Mongolian gerbils, and AKR/J mice. Larval development took place at the injection sites in gerbils and mice, but was delayed and abnormal. Some of the parasites in the injection site showed abnormal numerous budding. High pathogenicity was shown after subcutaneous and intraperitoneal injection as well as after oral inoculation.

KEY WORDS: *Taenia polyacantha,* rodents, migration route, larval development, abnormal development, histopathology, pathogenicity.

The larval *Taenia polyacantha* Leuckart, 1856, proliferates asexually and the metacestodes are found in the peritoneal and pleural cavities of rodents (Schiller, 1953; Rausch, 1959a; Murai and Tenora, 1973; Slais, 1973; Wiger et al., 1974; Tenora et al., 1979). Rausch and Fay (1988a) reported that the migration of postoncospheral stages was via the portal vein to the liver, and that initial larval development and formation of the primary vesicle took place in the liver. The production of secondary vesicles occurred after migration to the peritoneal cavity. Fujita et al. (1990) reported on the susceptibility and mortality of various small mammals to infection by *T. polyacantha* after oral inoculation of eggs. Red-backed voles and cotton rats showed high mortality at the early phase of infection. In those hosts, the sites of early larval development and the production of primary and secondary vesicles were different from those reported by Rausch and Fay (1988a).

In this study we examined the route of migration and development of the larvae, and assessed pathological changes in red-backed voles and Mongolian gerbils. In order to determine whether the postoncospheral development of *T. polyacantha* is pathogenic, oncospheres were inoculated parenterally in red-backed voles, AKR/J mice, and Mongolian gerbils.

Materials and Methods

CESTODE: Metacestodes were isolated from northern voles, *Microtus oeconomus,* trapped at Savoonga, St. Lawrence Island, Alaska, in 1988 and 1989. In the laboratory, they were then transplanted surgically into the peritoneal cavity of Mongolian gerbils, *Meriones unguiculatus,* which were maintained for 3 months. Eight mature metacestodes were then administered orally to a cestode-free mongrel dog. Gravid segments expelled in feces of the dog were collected daily and stored at 4°C in saline containing penicillin, streptomycin, and fungizone (Mitchell et al., 1977; Williams et al., 1981). Eggs from the gravid segments were used within 1 month of collection.

EXPERIMENTAL ANIMALS: Mongolian gerbils and AKR/J inbred mice were bred and maintained in our laboratory. Red-backed voles, *Clethrionomys rufocanus bedfordiae,* were trapped in a shelter-belt near Sapporo City, Hokkaido, and maintained in our laboratory at 22–24°C.

EXPERIMENT 1 (observations on early larval migration): Red-backed voles (18 ♂; age not determined) and Mongolian gerbils (15 ♂, 9 ♀; 4–8 wk old) were used. Individual hosts were inoculated with 5,000 to 100,000 eggs by stomach-tube under light ether anesthesia, and killed by exsanguination under ether anesthesia at 12 hr postinfection (PI) and at 24-hr intervals up to 6 days PI; animals that died were examined at different intervals. Washings from the peritoneal

cavity were examined for larval cestodes by means of a dissecting microscope (at 35×). The recovered larvae were fixed in 10% formalin and mounted in glycerin jelly. The small intestine was divided into 5 equal parts, and the middle portion of each, and all abdominal and thoracic organs, were fixed in 10% buffered neutral formalin and embedded in paraffin by standard methods. Paraffin sections, cut at 4–6 μm, were stained with hematoxylin-eosin (HE) and Alcian blue-periodic acid-Schiff (AB-PAS) for microscopic examination. Sixteen to 20 serial sections of organs in which larval cestodes were found were studied, and the maximal lengths and widths of the vesicles were recorded. For electron microscopy, the small intestines of rodents of both species were fixed on day 5 PI in 3.0% glutaraldehyde in 0.1 M cacodylate buffer, and then postfixed in 1.0% osmium tetroxide in 0.1 M cacodylate buffer. The segments of the intestines were dehydrated in graded ethanol and embedded in Quetol-812. Ultrathin sections were cut and stained with uranyl acetate and lead citrate, and subsequently observed and photographed with an HITACHI HU-12A electron microscope.

EXPERIMENT 2 (parenteral injection of oncospheres): Mongolian gerbils (6 ♂, 18 ♀; 6–8 wk old), AKR/J mice (8 ♂, 7 ♀; 4 wk old), and red-backed voles (5 ♂, 3 ♀; age not determined) were used. The experimental design is shown in Table 1. The oncospheres were hatched with artificial intestinal fluid (Silverman, 1954); 1,000 or 20,000 oncospheres were injected subcutaneously or intraperitoneally in sterile saline. Individual animals were killed by exsanguination on days 19 and 21 PI and autopsied immediately after death. Sites of injection were washed with saline and examined for presence of vesicles using a dissecting microscope. Vesicles recovered were fixed in 10% formalin and stained with Schneider's aceto-carmine for morphological examination. If gross lesions were observed in the tissues, sections were examined microscopically.

To compare larval development in the subcutaneous tissue and in the peritoneal cavity, and to define relative pathogenicity, Mongolian gerbils also were inoculated orally with 300 or 500 eggs.

Results

Experiment 1

Early larval development was discerned only in the wall of the small intestine and in the mesenteric lymph nodes in voles and gerbils. Average size of the developing larvae and their locations are shown in Table 2.

RED-BACKED VOLES: Developing larvae were found in the lamina propria of the small intestine between 12 hr PI and 5 days PI. They were present in the submucosa or passing through the walls of the lymphatic lacteal on the third day PI (Figs. 1, 2). On day 4 PI, they had penetrated further and were recovered mainly in the submucosa and muscularis, as well as having reached the afferent lymph vessels and the peripheral and marginal sinuses of the mesenteric lymph nodes. After 5 days PI, some larvae were found in the peritoneal cavity, and from the sixth day PI they were no longer observed in the wall of the small intestine or mesenteric lymph nodes.

Table 1. The number of eggs or oncospheres of *Taenia polyacantha* in each inoculation route.

	Dose of inoculum	No. of hosts examined
Oral inoculation (shelled eggs)		
Mongolian gerbils	300	6
	500	6
Subcutaneous injection (oncospheres*)		
Mongolian gerbils	1,000	4
AKR/J inbred mice	1,000	8
Red-backed voles	20,000	4
Intraperitoneal injection (oncospheres*)		
Mongolian gerbils	1,000	6
AKR/J inbred mice	1,000	7
Red-backed voles	20,000	4

* Oncospheres were hatched artificially.

By 2 days PI, the early vesicle contained several prominent nuclei and PAS-positive granules, and was always surrounded by a halo-like, amorphous area, about 4–8 μm in width, within which was AB-positive material. Concurrent with a great increase in size of larva between days 2 and 3 PI, the amorphous area faded out almost completely. At this time, marked cellular differentiation occurred; a central cavity began to develop and the primary vesicle was formed. A few primary vesicles with buds were recovered in the wall of the small intestine on day 5 PI. On day 6 PI, the vesicles in the peritoneal cavity had produced some buds, and numerous host cells covered their surfaces (Fig. 3).

Between days 2 and 3 PI, slight hemorrhage and infiltration by inflammatory cells, mainly neutrophils and mononuclear cells, were observed in the wall of the small intestine and, at this time, blood-tinged ascitic fluid began to accumulate. These changes, especially extensive hemorrhage and purulent inflammation mainly surrounding the vesicles, became prominent in the wall of the small intestine, and thereafter in the mesenteric lymph nodes (Figs. 4, 5). Electron microscopically, it was determined that numerous neutrophils adhered closely to the surface of the tegument of the vesicles, and the number of cytoplasmic granules decreased (Fig. 6). The volume of ascitic fluid increased. After 4 days PI, inflammatory cells, mainly neutrophils accumulated in the mesenteric lymph nodes and

Table 2. Size and location of early larval stage of *Taenia polyacantha* in gerbils and voles.

Host		Days postinfection			
		0.5	1	2	3
Gerbil	Size (μm)*	21.0 × 14.8	22.2 × 17.8	38.0 × 25.2	61.5 × 35.5
	Location**	SI (LP) MLN	SI (LP) MLN	SI (LP) MLN	SI (LP ~ SM) MLN
Vole	Size (μm)*	22.2 × 14.6	23.8 × 16.3	36.8 × 27.8	85.7 × 62.8
	Location**	SI (LP)	SI (LP)	SI (LP)	SI (LP ~ SM)

* Average maximum length and width of each parasite measured in histological sections.
** SI = small intestine; LP = lamina propria; SM = submucosa; M = muscularis; MLN = mesenteric lymph node; PC = peritoneal cavity.
*** External budding.

spleen, along with hemorrhage, and the number of lymphocytes, decreased in these lymphatic organs.

MONGOLIAN GERBILS: By 3 days PI, developing larvae were recovered in the lamina propria and submucosa of the small intestine, especially in the jejunum and ileum. After 12 hr PI, they were found in the afferent lymph vessels and marginal sinuses of the mesenteric lymph nodes. On day 6 PI, vesicles were found in the peritoneal cavity.

The early larval development in gerbils was similar to that in the red-backed voles, with the larvae showing great enlargement between days 2 and 3 PI, and with the beginning development of a central cavity and primary vesicles. On day 6 PI, vesicles recovered from the peritoneal cavity displayed some buds.

Microscopically, a slight infiltration by inflammatory cells was evident in the lamina propria and submucosa of the small intestine after day 1 PI. Small accumulations of inflammatory cells were scattered focally in the dilated marginal sinus and capsule of the mesenteric lymph nodes, especially around the larvae, between days 4 and 6 PI (Fig. 7). A slight accumulation of turbid ascitic fluid with fibrin occurred on day 5. After 4 days, a few neutrophils adhered to the surfaces of the vesicles, and on day 5, their pseudopods were loosely attached to the fragments of microvilli (Fig. 8).

Experiment 2

Almost all of the Mongolian gerbils inoculated orally with 300 and 500 eggs were alive until the scheduled date of autopsy, but almost all those injected parenterally with 1,000 and 20,000 oncospheres died before 21 days PI. Development and pathological changes associated with the routes of inoculation are described below.

Oral inoculation

MONGOLIAN GERBILS: Ten to 58 ($\bar{x} = 31$) secondary vesicles were recovered from the peritoneal cavity in 5 of 12 infected gerbils necropsied on days 19 and 21 PI. Most of the larvae on day 19, ranging from 1.8 to 2.4 mm in length and 1.6 to 1.8 mm in diameter, had developed rostellar cones, and about half of them exhibited 2 rows of developing rostellar hooks (Fig. 9). Macroscopically, the infected gerbils exhibited a slight accumulation of turbid ascitic fluid, with fibrous adhesions on the serosa of abdominal organs.

Subcutaneous injection

MONGOLIAN GERBILS: Larval cestodes were recovered from the injection site of 3 of 4 gerbils that died between 12 and 19 days PI. Most of those by day 19 PI had formed single secondary vesicles, 88–272 μm in length and 80–152 μm in diameter; some still consisted of aggregations of secondary vesicles with less than 35 buds, and ranged from 368 to 552 μm in length by 208 to 360 μm in diameter (Fig. 10). An accumulation of nuclei was observed at the distal ends of detached secondary vesicles. At the site of injection, marked hemorrhage and edema were noted, and microscopically extensive necrosis and infiltration by eosinophils and neutrophils were observed in the dermis, subcutaneous tissues, and underlying skeletal muscles.

AKR/J MICE: Four mice died between 9 and 19 days PI. Vesicles were recovered from the injection sites of 6 of 8 animals. On days 9 and 11 PI all had formed single vesicles, apparently

Table 2. Continued.

Days postinfection		
4	5	6
99.0 × 54.6 MLN	123.4 × 65.0 MLN	990 × 340*** MLN PC
100.6 × 65.6 SI (LP ~ M), MLN	159.2 × 80.8*** SI (LP ~ M), MLN, PC	335 × 212*** PC

a primary vesicle, and most of those on days 19 and 21 PI had abnormally developed aggregations of secondary vesicles, with 32 to more than 50 buds; the aggregations ranged from 440 to 1,180 μm in length, and 200 to 980 μm in diameter. Nuclei had accumulated at the distal ends of almost all secondary vesicles, and a few were developing invaginated canals (Fig. 11). Gross lesions at sites of injection were similar to those in gerbils: extensive necrosis, hemorrhage, and a slight accumulation of inflammatory cells between the epidermis and underlying skeletal muscles were observed microscopically.

RED-BACKED VOLES: Two of 4 infected voles died between 5 and 8 days PI. They exhibited mild ascites with blood; extensive edema and slight necrosis were observed in the subcutaneous tissue. Larval cestodes were found in only 1 vole, which died on day 5 PI. In that animal, numerous ecchymoses were present on the serosa of the small intestine. Microscopically, the larvae consisted of primary vesicles ranging from 60 to 112 μm in length, and 40 to 64 μm in diameter. They were found mainly in the hemorrhagic lesions between the lamina propria and the muscularis. Slight hemorrhage and focal degenerative changes also occurred in the mesenteric lymph nodes.

Intraperitoneal injection

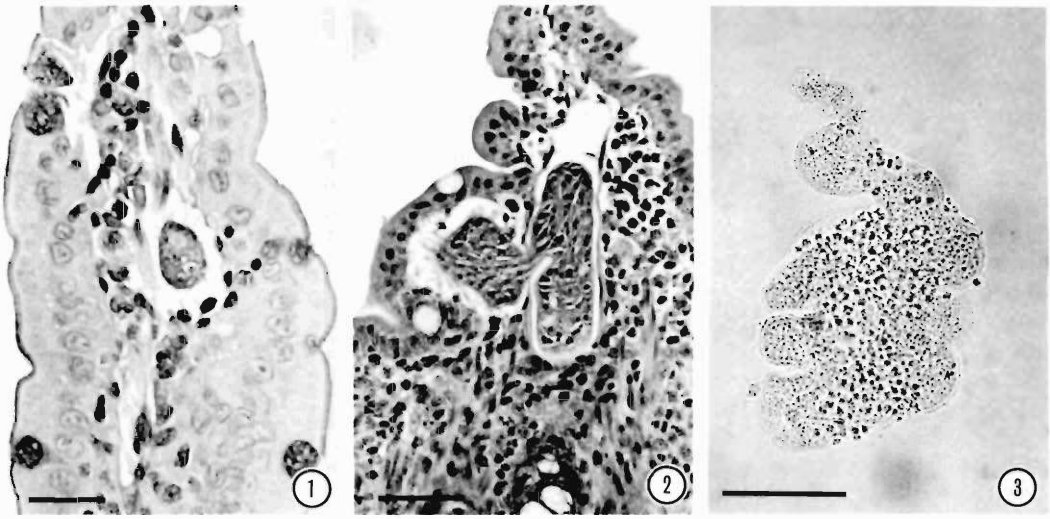
MONGOLIAN GERBILS: All gerbils died between 15 and 17 days PI, and larval cestodes were recovered from the peritoneal cavities. Almost all of the larval cestodes on day 17 formed single primary vesicles or 2 secondary vesicles, which were rounded and subspherical in form, and ranged from 112 to 416 μm in length and 96 to 224 μm in diameter. Some had formed abnormally minute aggregations of secondary vesicles, with 16 to more than 50 buds, which ranged from 384 to 584 μm by 240 to 442 μm in diameter; their surfaces were covered with numerous host cells (Fig. 12). Macroscopically, slight yellowish or turbid ascitic fluid was present, and all abdominal organs were congested.

AKR/J INBRED MICE: Six of the 7 infected mice died between 11 and 15 days PI; the last was necropsied on day 21 PI. Developing larvae were recovered from the peritoneal cavities of 6 mice. By day 15, the larvae had formed single or numerous secondary vesicles, all of similar size, with the aggregations ranging from 384 to 1,096 μm in length and 252 to 704 μm in diameter. Accumulations of nuclei at the distal ends of the vesicles had formed on day 12 PI. On day 21 PI, about half of the vesicles had detached, and some had developed invaginated canals which ranged from 184 to 216 μm in length by 104 to 128 μm in diameter.

RED-BACKED VOLES: Three of the 4 infected voles died by the seventh day PI. They exhibited mild ascites, with the fluid stained by blood, and all of the abdominal organs were congested. Slight hemorrhages had occurred in the mesenteric lymph nodes, but no larvae could be found microscopically in those lesions. Primary vesicles with central cavities were found under the phrenic serosa of the liver in only 1 vole dead on day 5 PI; they ranged from 136 to 200 μm in length by 72 to 112 μm in diameter. Focal accumulations of inflammatory cells and necrosis surrounded the larvae (Fig. 13). In the hepatic lymph nodes, a decrease in the number of lymphocytes had occurred, caused by focal degenerative change and hemorrhage.

Discussion

After the eggs of taeniids are ingested, the oncospheres hatch and become activated in the small intestine, after which they penetrate into the mucosal epithelium. The usual route of migration of the oncospheres is via the portal vein or lymphatic vessels. For example, it is well known that the oncospheres of *Taenia hydatigena, T. taeniaeformis, T. pisiformis,* and *Echinococcus multilocularis* migrate to the liver via the portal vein (Olsen, 1974). Concerning *T. polyacantha* in *Microtus oeconomus,* Rausch and Fay (1988a) reported that oncospheres migrate from the small intestine to the liver via the portal vein, and that early larval development takes place in the liver. Furthermore, Dorosz (1968) and Tenora et al. (1988) reported that metacestodes of *T. polyacantha* are localized under the



Figures 1–3. Larval *Taenia polyacantha* in red-backed voles. 1. Oncospheres situated in the lamina propria of the small intestine at 12 hr PI. AB-PAS stain. Scale bar = 20 μm. 2. The embryo passing through the wall of the lymphatic lacteal from the lamina propria, day 3. HE stain. Scale bar = 50 μm. 3. Developing secondary vesicles in the peritoneal cavity, day 6. Scale bar = 100 μm.

hepatic serosa of naturally infected hosts. These reports suggest that the liver is the typical site of early development of the larval cestode. However, in the rodents used in the present study, early development of the larvae was found to take place in the wall of the small intestine and in the mesenteric lymph nodes. Fujita et al. (1990) reported that larval *T. polyacantha* in cotton rats, *Sigmodon hispidus,* were recovered from the same organs as in gerbils and red-backed voles on days 8 and 10 PI. It is suggested that the oncospheres had a predilection for the wall of the small intestine and the mesenteric lymph nodes, and that those sites were peculiar to *T. polyacantha.* Since a few larvae were recovered from the liver of a red-backed vole inoculated intraperitoneally, the liver of that rodent appears also to be a site of development of the metacestode. Many larvae were situated in the afferent lymph vessels and marginal sinus of the mesenteric lymph nodes in gerbils and red-backed voles. We considered that the larvae could not migrate farther via the lymphatic system, because their size was too great relative to the diameter of the lymph vessels. The migration route of oncospheres in the present study was different from that reported by Rausch and Fay (1988a) in *Microtus oeconomus.* The isolates of *T. polyacantha* used in both investigations were evidently the same, having been obtained from voles, *M. oeconomus,* collected on St. Lawrence Island, in the Bering Sea. The differences in migration routes of the oncospheres appear to be related to differences in species of rodents used as experimental intermediate hosts.

Budding of the primary vesicles in the wall of the small intestine and in the mesenteric lymph nodes was observed by us from day 5 PI. Rausch and Fay (1988a) suggested that budding occurred after the early-stage vesicles had migrated from the liver into the peritoneal cavity; they reported that the larvae in the liver on day 5 PI were only

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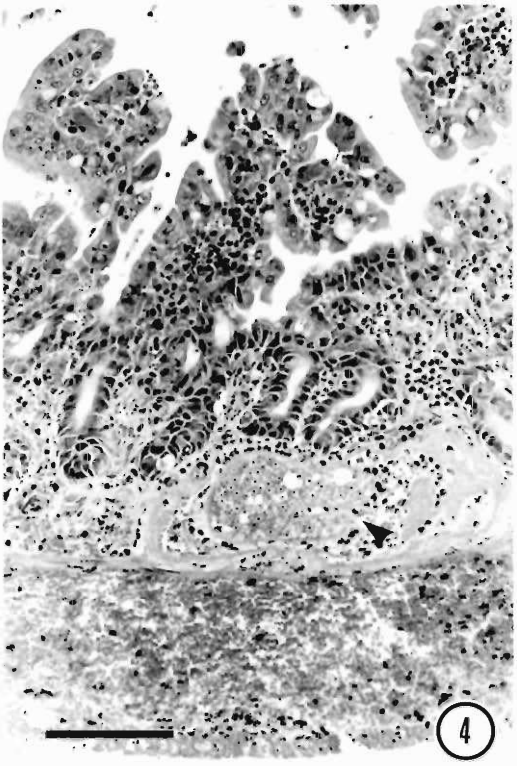
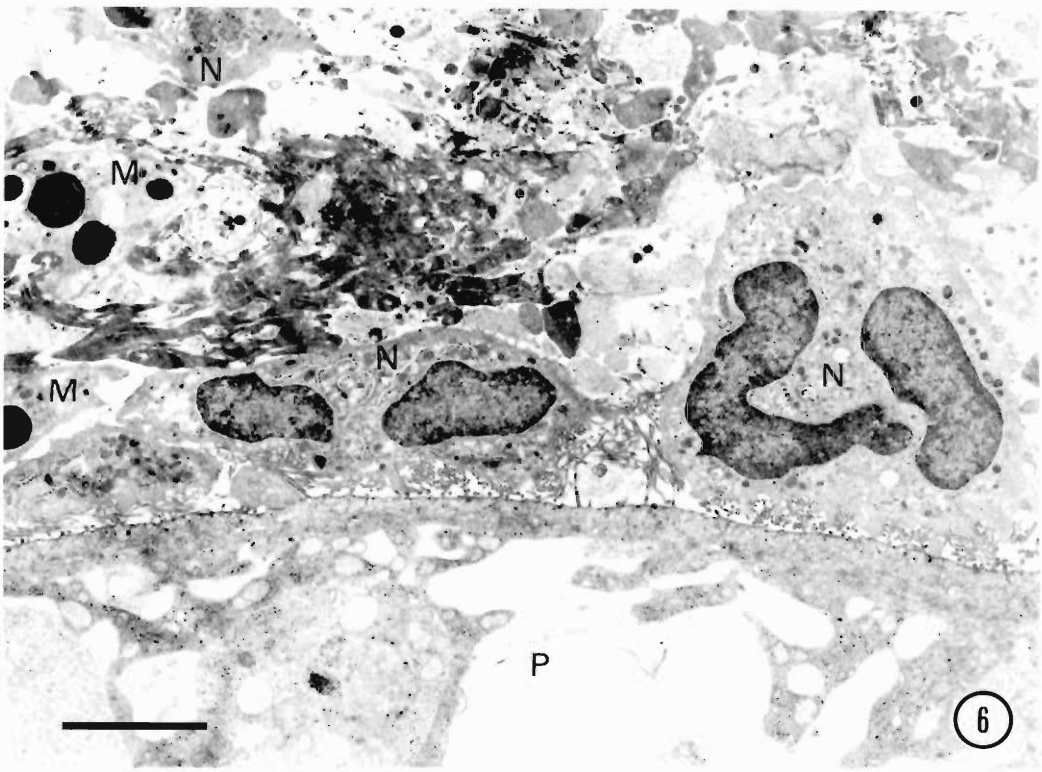
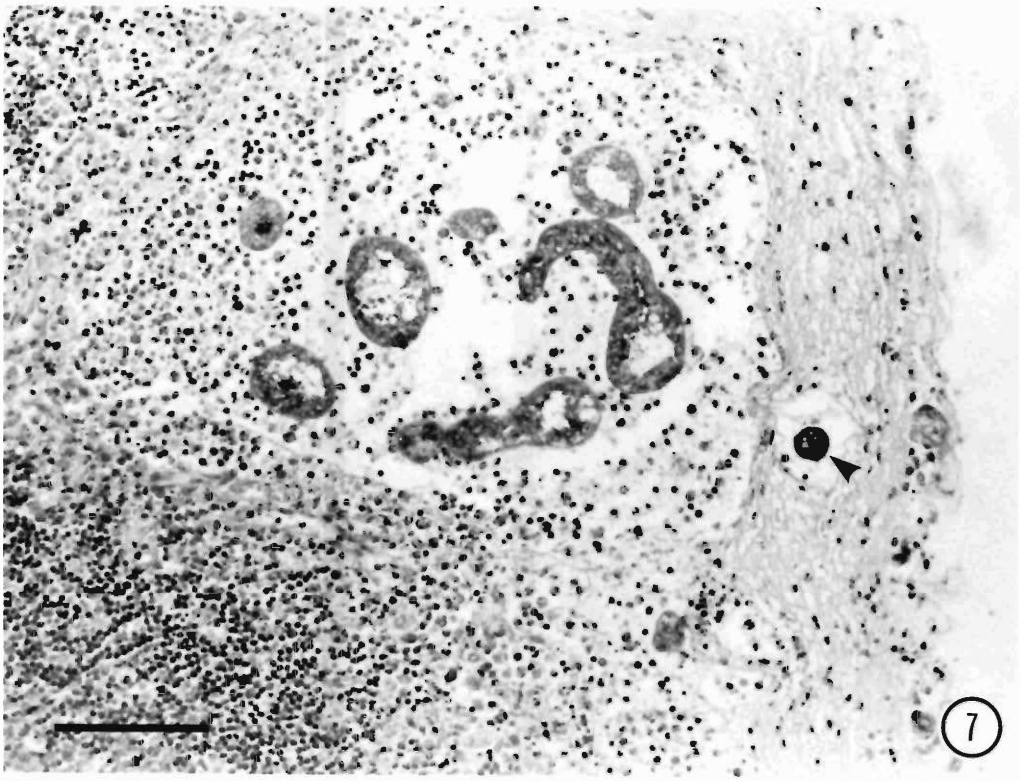
Figures 4–6. Pathological changes caused by larval *Taenia polyacantha* in the red-backed vole, day 5 PI. 4. Extensive hemorrhage and accumulation of numerous inflammatory cells surrounding the budding larva (arrowhead) in the wall of the small intestine. HE stain. Scale bar = 100 μm. 5. Marked decrease in the number of lymphocytes caused by extensive hemorrhage and degenerative changes in tissues around the larval cestode (arrowhead) in the mesenteric lymph node. AB-PAS stain. Scale bar = 100 μm. 6. Transmission electron micrograph of the larval cestode in the injured muscularis of the small intestine. Numerous neutrophils (N) and macrophages (M) closely attached to the surface of the vesicle (P). Scale bar = 5 μm.







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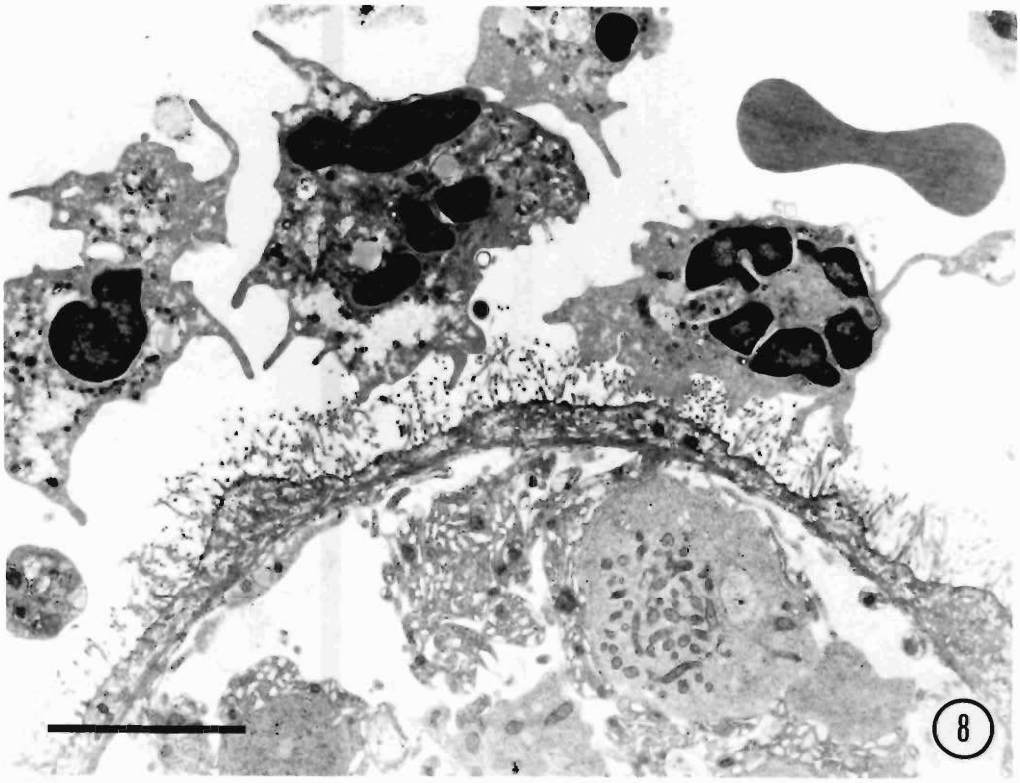

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19 and 22 μm in greater diameter. In the present study, the average size of larvae on day 5 PI was 123 μm in length and 65 μm in diameter; thus, they were much larger than those described by Rausch and Fay (1988a).

Although the early development of the larval cestode was quite similar in gerbils and red-backed voles, it produced different pathological changes in the 2 hosts. In the gerbils, the tissue-response was slight, but in red-backed voles, marked purulent inflammation occurred, and numerous neutrophils attached closely to the tegumental surface of the larvae. Such changes in red-backed voles were not proportional to the number of eggs inoculated. Rausch and Fay (1988b) suggested that infections involving more than 5 eggs might be fatal, because of the inflammatory response evoked by the migration of the vesicles into the peritoneal cavity. The pathogenicity of other taeniid cestodes, i.e., *T. twitchelli* and *T. mustelae* is proportional to the number of eggs inoculated and the lesions caused by migrations of the larvae (Freeman, 1956; Rausch, 1959b). We considered from the present results that the high degree of pathogenicity and the high mortality in red-backed voles might be due to the greater susceptibility of that host, and marked pathological changes caused by the development and migrations of the larvae were the main cause of death. Since larvae developing in the subcutaneous tissues also were quite pathogenic, they possibly secrete some deleterious substances. Shield et al. (1973) reported that 8-day larvae of *T. pisiformis* exhibited PAS-positive secretory cells of large size, which had some effect in the migration of the larvae. In the larval stage of *T. polyacantha,* numerous PAS-positive cells of small size were observed, but the larger cells described by Shield were not observed. The mechanisms of pathogenesis in the case of *T. polyacantha* remain unclear.

When oncospheres of *T. hydatigena, T. saginata, T. ovis,* and *Echinococcus granulosus* were inoculated subcutaneously and intraperitoneally into the intermediate hosts, development of the larvae took place at the site of inoculation (Gemmell, 1962; Williams and Colli, 1970; Slais and Machnicka, 1976). In the present investigation, the development of the larval *T. polyacantha* took place at the injection sites in Mongolian gerbils and AKR/J mice, and involved the abnormal budding of numerous vesicles of small size. In *M. oeconomus,* fewer than 16 buds were usually produced (Rausch and Fay, 1988a). Parenteral inoculation of oncospheres may deprive the larva of appropriate stimuli and nutrients for normal development. A few larvae recovered from the peritoneal cavity of the intraperitoneally inoculated AKR/J mouse, however, were similar on day 21 PI to those from orally inoc-

Figure 9. Advanced stage of secondary vesicle, showing the rostellar cone (arrow) and developing hooks, day 19 PI, from a Mongolian gerbil inoculated orally. Schneider's aceto-carmine stain. Scale bar = 100 μm.

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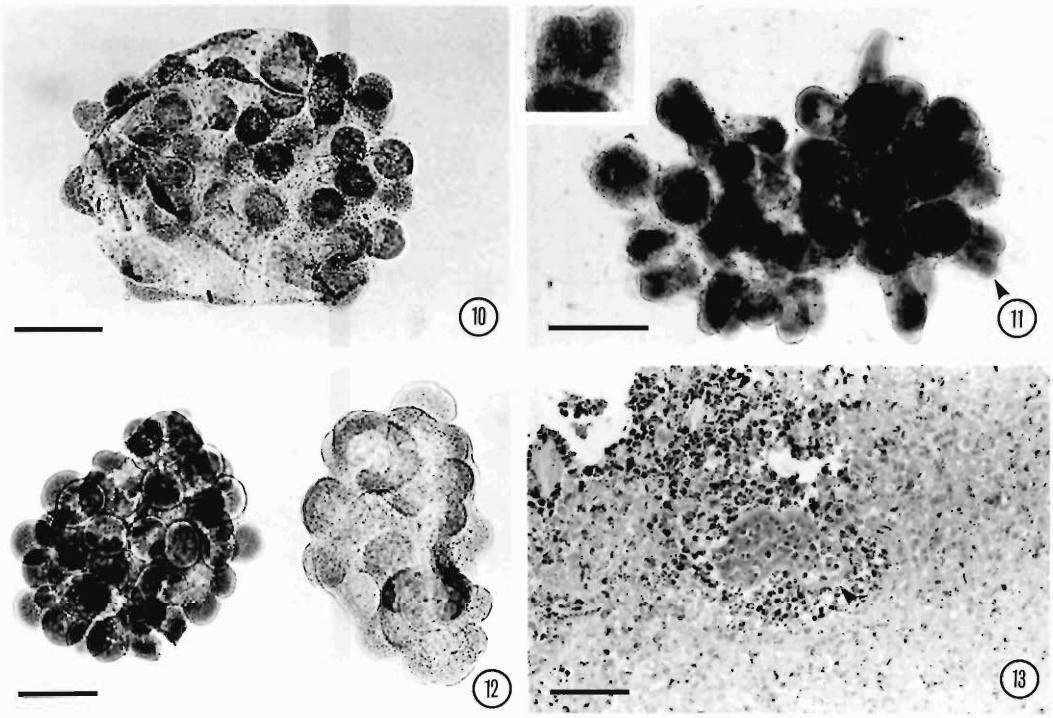
Figures 7, 8. Larval *Taenia polyacantha* in the mesenteric lymph node of Mongolian gerbils, day 5 PI. 7. Focal accumulation of inflammatory cells surrounding the vesicles in the marginal sinus and capsule. Vesicle in the afferent lymph vessel (arrowhead). AB-PAS stain. Scale bar = 100 μm. 8. Transmission electron micrograph of the vesicle in the marginal sinus. A few neutrophils cover the surface of the vesicle, and their pseudopods extend to fragments of the microvilli. Scale bar = 5 μm.



Figures 10–13. Larval *Taenia polyacantha* from parenterally inoculated rodents. 10. Proliferation of minute secondary vesicles, subcutaneous tissue of a Mongolian gerbil on day 19 PI. Schneider's aceto-carmine stain. Scale bar = 50 μm. 11. Vesicle with numerous buds, subcutaneous tissue of AKR/J mouse, day 19 PI. Schneider's aceto-carmine stain. Scale bar = 30 μm. Inset shows early formation of an invaginated canal in a secondary vesicle (arrowhead). 12. Proliferation of secondary vesicles, peritoneal cavity of a Mongolian gerbil, day 17 PI. Schneider's aceto-carmine stain. Scale bar = 50 μm. 13. Accumulation of inflammatory cells, with focal necrosis, around an early vesicle (arrowhead), beneath the phrenic serosa of the liver of a red-backed vole that died on day 5 PI. HE stain. Scale bar = 50 μm.

ulated hosts (Fujita et al., 1990). Many larvae were recovered in the wall of the small intestine of a red-backed vole that had been inoculated subcutaneously. This finding cannot now be explained, but the possibility of the migration of the oncospheres cannot be excluded.

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Literature Cited

Dorosz, J. 1968. Helminth parasites of small rodents living in areas irrigated by urban sewage of Wroclaw. Acta Parasitologica Polonica 16:375–396.

Freeman, R. S. 1956. Life history studies on *Taenia mustelae* Gmelin, 1790 and the taxonomy of certain taenioid cestodes from Mustelidae. Canadian Journal of Zoology 34:219–242.

Fujita, O., Y. Oku, H. K. Ooi, M. Kamiya, and R. L. Rausch. 1990. Susceptibility of rodents to *Taenia polyacantha* and their mortality along the course of infection. Japanese Journal of Parasitology 39: 284–289.

Gemmell, M. A. 1962. Natural and acquired immunity factors inhibiting penetration of some hexacanth embryos through the intestinal barrier. Nature (London) 194:701–702.

Mitchell, G. F., J. W. Goding, and M. D. Rickard. 1977. Studies on immune responses to larval cestodes in mice. Increased susceptibility of certain mouse strains and hypothymic mice to *Taenia taeniaeformis* and analysis of passive transfer of resistance with serum. Australian Journal of Experimental Biology and Medical Science 55:165–186.

Murai, E., and F. Tenora. 1973. Some taeniid species (Cestoidea) parasitizing vertebrates (Rodentia,

Carnivora, Strigiformes) in Hungary. Acta Zoologica Academiae Scientiarum Hungarica 19:125–132.

Olsen, O. W. 1974. Animal Parasites. Their Biology and Ecology, 3rd ed. University Park Press, Baltimore. 525 pp.

Rausch, R. L. 1959a. Studies on the helminth fauna of Alaska. XXXV. On the identity of certain cestodes (Taeniidae) from foxes. Proceedings of the Helminthological Society of Washington 26:125–131.

———. 1959b. Studies on the helminth fauna of Alaska. XXXVI. Parasites of the wolverine, *Gulo gulo* L., with observations on the biology of *Taenia twitchelli* Schwartz, 1924. Journal of Parasitology 45:465–484.

———, **and F. H. Fay.** 1988a. Postoncospheral development and cycle of *Taenia polyacantha* Leuckart, 1856 (Cestoda: Taeniidae). First part. Annales de Parasitologie Humaine et Comparée 63:263–277.

———, **and** ———. 1988b. Postoncospheral development and cycle of *Taenia polyacantha* Leuckart, 1856 (Cestoda: Taeniidae). Second part. Annales de Parasitologie Humaine et Comparée 63:334–348.

Schiller, E. L. 1953. Studies on the helminth fauna of Alaska. XV. Some notes on the cysticercus of *Taenia polyacantha* Leuckart, 1856, from a vole (*Microtus oeconomus operarius* Nelson). Journal of Parasitology 39:344–347.

Silverman, P. H. 1954. Studies on the biology of some tapeworms of genus *Taenia*. I. Factors affecting hatching and activation of taenid ova, and some criteria of their viability. Annals of Tropical Medicine and Parasitology 48:207–215.

Shield, J. M., D. D. Heath, and J. D. Smyth. 1973. Light microscope studies on the early development of *Taenia pisiformis* cysticerci. International Journal for Parasitology 3:471–480.

Slais, J. 1973. Functional morphology of cestode larvae. Pages 395–480 *in* B. Dawes, ed. Advances in Parasitology. Vol. 11. Academic Press, London.

———, **and B. Machnicka.** 1976. Appearance of a temporary hook anlage in the early development of *Taenia saginata* Goeze, 1782. Zoologischer Anzeiger (Leipzig) 196:85–92.

Tenora, F., R. Wiger, and V. Barus. 1979. Seasonal and annual variations in the prevalence of helminths in a cyclic population of *Clethrionomys glareolus*. Holarctic Ecology 2:176–181.

———, **L. Beranek, and M. Stanek.** 1988. Larvocysts of the cestode *T. polyacantha* (Leuckart, 1856) parasitizing *Oryctolagus cuniculus*. Folia Parasitologica (Praha) 35:21–22.

Wiger, R., L. Lien, and F. Tenora. 1974. Studies on the helminth fauna of Norway. XXXIII. *Tetratirotaenia polyacantha* (Leuckart, 1856) Abuladze 1964, a parasite of *Clethrionomys glareolus* in Norway. Norwegian Journal of Zoology 22:61–64.

Williams, J. F., A. M. Shearer, and M. M. Ravitch. 1981. Differences in susceptibility of rat strains to experimental infection with *Taenia taeniaeformis*. Journal of Parasitology 67:540–547.

———, **and C. W. Colli.** 1970. Primary cystic infection with *Echinococcus granulosus* and *Taenia hydatigena* in *Meriones unguiculatus*. Journal of Parasitology 56:509–513.